# **U.S. Department of Labor**

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**Issue Date: 29 August 2007** 

Case No.: 2003-BLA-00171

BRB No.: 06-0440 BLA

In the Matter of:

R. C.

Claimant

v.

#### BLASCHAK COAL CORPORATION

Employer

and

# ROCKWOOD CASUALTY INSURANCE COMPANY

Carrier

and

# DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS,

Party-in-Interest

Appearances:

Helen M. Koschoff, Esquire

for Claimant

Christopher J. Wildfire, Esquire

for Employer & Carrier

Before: **RALPH A. ROMANO** 

**Administrative Law Judge** 

# <u>DECISION AND ORDER ON REMAND – DENIAL OF BENEFITS</u>

This matter is before me on remand from the Benefits Review Board (the Board). In its Decision and Order dated December 29, 2006, the Board vacated the finding I rendered in my February 1, 2006 Decision and Order on Remand, that the existence of complicated pneumoconiosis had been established. The Board held that my finding that Drs. Ahmed,

Cappiello and Miller did not positively attribute the large opacities they found on x-ray readings to pneumoconiosis precluded Claimant's entitlement to the Section 718.304 presumption as a matter of law. In discussing my review of the x-ray evidence, the Board affirmed my finding that that evidence was sufficient to establish the existence of pneumoconiosis pursuant to 20 C.F.R. § 718.202(a)(1). The Board found error however, in the weighing of the evidence under 20 C.F.R. § 718.202(a) and remanded this matter for reconsideration of this issue. The Board directed that after discussing and weighing all of the relevant x-ray and medical opinion evidence, I must determine whether Claimant had established the existence of pneumoconiosis pursuant to 20 C.F.R. § 718.202(a), and if so, then the evidence must be reviewed in order to determine whether total disability due to the disease had been established. If benefits were awarded, the grounds for the modification must also be determined, i.e., whether it was due to a mistake in a determination of fact or a change in conditions, given that this finding determined the date from which benefits would commence.

On remand, a Briefing Schedule Order was issued, providing all parties the opportunity to submit briefs on or before thirty days from the date of the Order. Employer submitted a brief on May 9, 2007.

To briefly recap, this matter has been before me on several occasions. In my Decision and Order dated April 13, 2000, I held that the Miner had failed to establish the existence of pneumoconiosis or total disability due thereto, finding that the Miner was disabled due to his tuberculosis and polio. After a remand from the Board, the matter having been appealed, I issued a Decision and Order dated March 25, 2002, wherein benefits were again denied. Claimant filed a request for modification and on March 1, 2004, I issued a Decision and Order awarding benefits. In that decision, I found that a change in condition had been established. An appeal was filed and the Board remanded the matter, resulting in my Decision and Order dated February 1, 2006, and which is the subject of the Board's most recent remand.

After reviewing my Decision and Order, the Board directed that, on remand, the totality of the evidence must be weighed, in order to determine whether the evidence is sufficient to establish the existence of pneumoconiosis pursuant to 20 C.F.R. § 718.202(a). In particular, the Board pointed out that all the evidence submitted prior to the Claimant's modification request must be considered. The Board noted that in my 2004 Decision and Order, I did not consider the medical opinion evidence submitted prior to Claimant's request for Modification, namely the opinions of Drs. Galgon, Kaplan, R. Kraynak, Rashid, Renn, Patel and Ahluwalia, and that I erred in my consideration of the x-ray evidence submitted on modification. The issue of any error in consideration of the x-ray evidence made in the 2004 decision would appear to be moot, given that the Board affirmed my finding in my 2006 decision, that pneumoconiosis had been established pursuant to 20 C.F.R. § 718.202(a)(1).

Claimant has established, via x-ray evidence, the existence of clinical pneumoconiosis. This finding must be weighed against the contrary medical opinion. Clinical or medical pneumoconiosis, as determined by a review of the chest x-rays, is a disease of the lung characterized by certain opacities appearing on the chest x-ray. It is these opacities which I have found to have been established. When weighing this finding against the medical opinion evidence, having considered the reports of Drs. Levinson, Fino, Galgon, Kaplan, Rashid, Renn,

Patel, Kraynak and Ahluwalia, as well as the treatment records, I find that the x-ray evidence is, in fact, sufficient to establish that a pneumoconiosis is present. Thus, I find that the medical reports cannot overcome the finding of clinical pneumoconiosis made by several radiologists in this case, and more particularly, by the weight of the readings of the most recent x-ray of record. That medical evidence is set forth below.

The Miner was hospitalized on November 22, 1996 with a final diagnosis of acute coronary insufficiency. (DX 43). The record indicates that the Miner quit smoking eight months ago and previously smoked a pack of cigarettes for day for fifteen years. Dr. Joseph Weber noted the Miner's chief complaints as diaphoresis, severe vertigo and nausea. In a Consultation Report after examining the Miner on November 23, 1996, Dr. D. Patel listed a diagnosis of chronic obstructive pulmonary disease, right apical pleural disease, history of tuberculosis and presyncope. Dr. Patel recorded that the Miner did not appear to have any active pulmonary disease however he did have chronic pulmonary disease related to cigarette smoking, changes related to his old tuberculosis as well as some occupational pneumoconiotic type problems. A Cardiology Outpatient Discharge Summary from January 28, 1997 lists a discharge diagnosis of hypertensive cardiovascular disease and a secondary diagnosis which includes mild left ventricular dysfunction, history of hypertension, hypercholesterolemia, COPD and history of pulmonary tuberculosis. (DX 43). It is signed by Dr. John H. Chapman. Several pages of handwritten notes are included as is a Cardiac Catheritzation Report.

Dr. H. S. Ahluwalia examined the Miner on June 9, 1998. (DX 43). He recorded 25 years of coal mine employment and a smoking history of approximately 20 years, ten years in which the Miner smoked a pack of cigarettes per day and the other ten years probably half a pack per day, for a total of 15 pack years. Dr. Ahluwalia noted that the Miner gave a history of tuberculosis contracted in 1966, the Miner having been hospitalized for three months. Based upon his examination, Dr. Ahluwalia noted obvious wasting of the shoulder muscles on the right side and decreased respiratory excursion on the right side. Based upon his examination, which included the taking of a chest x-ray and pulmonary function testing, Dr. Ahluwalia found the Miner to be suffering from scarring, right apex, with pulmonary fibrosis due to old, healed tuberculosis and reversible bronchospasm.

Dr. A. Rashid examined the Miner on October 22, 1998. (DX 12). A smoking history of one pack per day for fifteen years was recorded, the Miner having quit smoking in 1994. Based upon his examination, which included the taking of a chest x-ray (negative), pulmonary function testing and an EKG (abnormal), Dr. Rashid diagnosed coronary artery disease, hypertension and high cholesterol.

Dr. John P. Galgon examined the Miner on July 7, 1999. (DX 43). He recorded that the Miner contracted tuberculosis in 1965 and was treated for one year. He smoked a half to one pack of cigarettes per day for fifteen years, quitting in 1994 and worked for five years as an underground coal miner, drove a trailer truck for twelve years on the strippings and hauled coal for eight years. The Miner had polio at the age of two years, involving his right arm and right chest. Based upon his examination, which included the taking of a chest x-ray, pulmonary function and blood gas testing, as well as an electrocardiogram and oximetry, Dr. Galgon found a marked reduction in vital capacity with evidence of moderate to severe airways obstruction

secondary to old tuberculosis with marked retraction of the right upper lobe, resulting in hyperinflation as noted by x-ray. Dr. Galgon found that on x-ray densities were present, some or all of which "might also be secondary to old TB." In his opinion, the Miner's history of dyspnea was entirely consistent with the ventilatory disturbance measured and it was his finding that this disturbance was secondary to old tuberculosis and not secondary to the mild pneumoconiosis measured. In his opinion, the Miner did not have either impairment or disability due to coal worker's pneumoconiosis.

Dr. Galgon reviewed the Miner's medical records, submitting a report dated August 7, 1999. (DX 43). Based upon his review, Dr. Galgon found no reason to change his prior opinion, that the Miner did not have impairment or disability due to coal worker's pneumoconiosis. It was his opinion that the changes seen on x-ray represented prior tuberculosis and not coal worker's pneumoconiosis.

Dr. Peter Kaplan reviewed the medical evidence and read x-rays, submitting a report dated September 21, 1999. (DX 46). In his opinion, there was no evidence to suggest the presence of coal worker's pneumoconiosis or any functional impairment attributable to the Miner's occupational exposure to coal dust.

By report dated September 7, 1999, Dr. Raymond Kraynak stated that the Miner had been under his care since September 1, 1999. (DX 32). Five years of underground coal mine employment and 12.5 years of above ground coal mine employment were recorded. No smoking history was noted and with regard to the Miner's past medical history, Dr. Kraynak noted that the Miner had had the usual childhood illnesses and that his history was positive for polio as a child affecting the right side. He noted that the Miner was able to work without limitation relative to his polio. Based upon his examination, which included the taking of a pulmonary function study and chest x-ray read by Dr. Smith, Dr. Kraynak concluded that the Miner was totally and permanently disabled by his coal worker's pneumoconiosis. The office records of Dr. Kraynak have been submitted. (CX 2, 5). These records are handwritten, and for the most part, illegible. On several occasions, it is noted that the Miner was coming in for a "check-up for breathing."

The deposition testimony of Dr. Raymond Kraynak was taken on October 1, 1999. (DX 34). Dr. Kraynak testified that he is board-eligible in family medicine. According to Dr. Kraynak, the Miner had a minimal smoking history over a fifteen year period and he quit smoking in 1994. Based upon his examination, as well as his review of certain medical evidence Dr. Kraynak opined that the Miner suffered from coal worker's pneumoconiosis. He opined that the polio suffered by the Miner when he was two years of age had no significance on the Miner's current pulmonary condition. His tuberculosis also would not be a factor in his shortness of breath, while the coal worker's pneumoconiosis which was present is a progressive disease, meaning that the Miner could have developed it over time and it could get worse. In his opinion, it was more logical and more reasonable to conclude that coal worker's pneumoconiosis was the cause of his current respiratory problem, not the polio or the exposure to tuberculosis that was successfully treated almost thirty years ago. Dr. Kraynak also did not feel that the Miner's prior smoking history had an effect on his current pulmonary condition. Dr. Kraynak found it ridiculous to attribute the Miner's opacities on chest x-ray to his prior tuberculosis, as was done by Dr. Galgon. Dr. Kraynak disagreed with other findings rendered by Dr. Galgon with regard

to the etiology of the Miner's pulmonary condition. Dr. Kraynak opined that the Miner was totally and permanently disabled due to his coal worker's pneumoconiosis.

By report dated October 10, 1999, Dr. Joseph J. Renn, III stated that he had reviewed the medical evidence forwarded to him. (DX 47). Dr. Renn is board-certified in internal medicine, pulmonary disease and forensic medicine. Dr. Renn noted that he had before him five smoking histories ranging from 11 1/4 pack years to as much as 30 pack years. Based upon his review, Dr. Renn concluded that the Miner had old healed pulmonary tuberculosis, pulmonary emphysema, asthma, arteriosclerotic coronary vascular disease, systemic hypertension, hypercholesterolemia and poliomyelitis, none of which were contributed to by his exposure to coal mine dust. The pulmonary emphysema was the result of tobacco abuse and asthma is a disease of the general population.

Dr. John P. Simelaro submitted a report dated October 18, 1999, after his review of several x-ray readings, treatment records and medical reports and studies. (DX 39). Dr. Simelaro is board-certified in internal medicine and pulmonary disease. Based upon his review, he concluded that the Miner's tuberculosis did not cause his disability. In so concluding, Dr. Simelaro pointed to the fact that the Miner was appropriately treated after contracting tuberculosis in 1966, and he continued to work from 1967 to 1998. While his x-ray showed scars of tuberculosis in the right upper lobe, the right upper lobe "really does not contribute that much to lung function since its volumes are markedly decreased when compared to the lower lobes." It was his opinion that surgical removal or infectious destruction would not impact that much on lung function. As for the Miner's past history of polio, Dr. Simelaro found that the Miner's work record of heavy labor discounted any disabling neurological impairment. Therefore, it was his opinion that the Miner's moderate obstruction on pulmonary function testing had two possible etiologies: smoking or coal mine dust exposure. As he found the smoking history in this case to be mild (15 pack years quit in 1994), anthracosilicosis was what he concluded was the cause of the Miner's pulmonary compromise. It was his opinion that the Miner's coal dust exposure, dust in lungs, moderate obstructive pattern on PFT and smoking history all point to disability and there "can be no question" that coal worker's pneumoconiosis contributed significantly to these symptoms.

The deposition testimony of Dr. Galgon was taken on October 22, 1999. (DX 52). Dr. Galgon is board-certified in internal medicine and pulmonary disease. He is also a B-reader. Dr. Galgon testified to his examination of the Miner on July 7, 1999 and reiterated his opinion as stated in his report concerning that evaluation. According to Dr. Galgon, the Miner's polio, contracted when he was a toddler probably weakened the right side of his chest. There was decreased musculature on the right side of his chest consistent with his previous polio. The Miner also related to him that he used to smoke one half to one pack of cigarettes per day for about fifteen years, the Miner having quit smoking in 1994. Dr. Galgon also took note of the varying smoking histories in the medical reports and records and he had the opportunity to review medical records in this case. Dr. Galgon stated that the Miner's right arm was smaller than his left and there was marked limitation of movement of the right arm and decreased amount of muscle mass in the right arm. Dr. Galgon testified that his reading of a chest x-ray resulted in a reading of 1/0, which profusion was restricted to the left side. The Miner also had a volume loss on the right side, which in the opinion of Dr. Galgon, was due to the significant

tuberculosis the Miner had involving the right upper lung, as this caused the lung to become destroyed and volume loss to occur as a result. The lower lobe hyperinflates to take up the space and results in the changes seen in this Miner. In his opinion, there were no classifiable radiodensities on the right side and the densities seen "which we have to characterize as consistent with pneumoconiosis are almost surely secondary to old tuberculosis." Dr. Galgon stated that he could not tell the difference between tubercular opacities and pnemoconiotic opacities, however, the pattern in this case was more likely to be the pattern of tuberculosis than of pneumoconiosis. In his opinion, the Miner had both restrictive and obstructive lung disease.

Dr. Galgon opined that most of the reduction in the FVC seen in this case was the result of old tuberculosis. The obstructive defect could also be entirely secondary to the Miner's old tuberculosis, cigarette smoking being a likely aggravating factor. Blood gas testing and oximetry testing revealed that the Miner's oxygen saturation was normal. The testing established that the Miner could not have significant interstitial lung disease as a cause for his being short of breath, as even when he walked his oxygen saturation did not drop. Dr. Galgon's own examination and his review of records herein led him to opine that the Miner's marked reduction of vital capacity with evidence of moderate to severe airways obstruction was secondary to his old tuberculosis with marked retraction of the right upper lobe resulting in hyperinflation as noted by x-ray. "Cigarette smoking very likely is an added factor in this reduction in the vital capacity." While the opacities found on x-ray were consistent with pneumoconiosis, in his opinion, some or all of these rounded densities "might also be secondary to old tuberculosis." The records he reviewed also documented the presence of hypertension, hypercholesterolemia, mild left ventricular dysfunction, COPD and a history of pulmonary tuberculosis and polio. It was his opinion that the Miner did not have an impairment or disability due to coal worker's pneumoconiosis. He stated that the densities consistent with pneumoconiosis most likely represented old tuberculosis. Coronary disease and obstructive lung disease both contributed to the Miner's complaint of shortness of breath. Dr. Galgon recommended that the Miner not return to his prior work, due to his obstructive lung disease "that has nothing to do with coal worker's pneumoconiosis."

In a report dated November 21, 2002, Dr. Matthew Kraynak stated that the Miner had been under his care for several years. (DX 70). Dr. Kraynak recorded that the Miner had a history of polio, affecting the right side, a history of tuberculosis and a smoking history of fifteen pack years. Dr. Kraynak also reviewed the conflicting smoking histories reported by other physicians and the Miner's treatment records. Dr. Kraynak noted seventeen and a half years of coal mine employment. Based upon his examination, Dr. Kraynak found that the Miner was totally and permanently disabled secondary to coal worker's pneumoconiosis. Dr. Kraynak is board-certified in family medicine.

On January 15, 2003, Dr. Stephen Kruk examined the Miner because of significant and progressive shortness of breath. (CX 5). Seventeen years in the coal mines and a smoking history of fifteen pack years were recorded. Past medical history included polio at age two years and tuberculosis in 1962. Based upon his examination, which included the taking of a spirometry and the review of x-ray readings, Dr. Kruk found multiple factors to the Miner's progressive shortness of breath, including his coal mine employment, his tuberculosis, and his cigarette smoking. Dr. Kruk concluded that the Miner was totally and permanently disabled

secondary to his coal worker's pneumoconiosis as the primary culprit. Dr. Kruk is board-certified in internal medicine.

Dr. Sander J. Levinson examined the Claimant on May 1, 2003. (EX 5). Dr. Levinson also had the opportunity to review medical records prior to rendering his decision. Dr. Levinson recorded that the Miner had been hospitalized extensively for tuberculosis in 1966. He also had a history of polio at the age of two years, which affected his right arm and chest. Five and a half years of underground coal mine employment was recorded, as was a history of twelve years on the strippings. The Miner also indicated he worked for twelve years driving a truck in he strippings until he retired in 1998. A cigarette smoking history of one pack per day or less for fifteen years was listed, the Miner having not smoked in the past eight years. Based upon his examination, which included the taking of a chest x-ray, pulmonary function testing and blood gas studies, as well as a review of the evidence, Dr. Levinson concluded that the Miner suffered from old tuberculosis with considerable scarring and retraction of the right upper lobe with considerable marked loss in volume in the right lung. He stated that the fibrosis was secondary to old inactive previously treated pulmonary fibrosis. Dr. Levinson found evidence of obstructive airways disease with significant improvement post bronchodilator related to a prior history of cigarette smoking. He did not find that the Miner suffered any substantial disability or impairment as a result of his coal mine employment. Dr. Levinson is board-certified in internal medicine and pulmonary disease.

In a report dated July 9, 2003, Dr. Simelaro stated that he had reviewed additional evidence. (CX 6). His opinion remained that the Miner had pneumoconiosis along with cigarette smoking which had rendered him totally disabled. In a subsequent report dated September 17, 2003, Dr. Simelaro stated that he had reviewed Dr. Levinson's position. (CX 6). Dr. Simelaro stated that what was evident here was a classic case of tuberculo-anthraco-silicosis. He noted that the Miner worked to 65 years of age in construction. If tuberculosis caused the Miner's respiratory disability, then he would have been unable to work after his bout of tuberculosis in 1962, so it was apparent to him that something else caused the Miner's disability. Dr. Simelaro took into account the Miner's cigarette smoking and concluded that while Dr. Levinson found that improvement with inhaled bronchodilators would not be present if the Miner had coal worker's pneumoconiosis, in this case, there was bronchial destruction of tuberculosis and to some extent, cigarette smoking. So the Miner had some bronchospasms as do many other coal miners with this condition. It was his conclusion that the Miner was totally disabled as a result of coal mining and to a lesser extent cigarette smoking and tuberculosis.

Dr. David S. Prince reviewed the evidence of record by report dated July 22, 2003. (CX 6). It was his opinion that pneumoconiosis was a substantial contributing factor to the Miner's respiratory disability. The Miner's tuberculosis involving the right upper lobe had also clearly produced evidence of volume loss and fibrous scarring. However, even if the entire right upper lobe were non-functional, this would not produce the degree of impairment present in the Miner.

Dr. Galgon had the opportunity to review medical records, and submitted a report dated September 19, 2003. (EX 5). These records included x-ray readings, the reports and deposition testimony of Dr. Kraynak, blood gas studies, pulmonary function testing and the validation

reports concerning same, his own deposition testimony, and the testimony of the Miner. Dr. Galgon pointed out that the pulmonary function studies have varied through the years, but in his opinion, the apparent reduction in the FVC was secondary to the Miner's effort rather than to worsening of his condition. Based upon his review, Dr. Galgon opined that the Miner had not developed pneumoconiosis since my previous decisions, further concluding that there was no impairment or disability due to pneumoconiosis.

Dr. Levinson submitted a follow-up report dated September 21, 2003. (EX 5). Therein, he stated that he had had the opportunity to review additional evidence, including chest x-rays. Dr. Levinson found that the fibrosis that the Miner suffered was secondary to old inactive previously treated pulmonary tuberculosis with evidence of obstructive airways disease showing significant improvement post bronchodilator. He concluded that these conditions were not in any way related to coal mine employment. Dr. Levinson stated that he found no evidence that the Miner had developed any form of pneumoconiosis or pulmonary disability related to his prior inhalation of coal dust or any form of pulmonary disability related to his previous coal mine employment. His symptomatology was "clearly related to the residuals of old tuberculosis with scarring as well as cigarette smoking induced obstructive lung disease and the effects of previous polio myelitis."

By report dated September 26, 2003, Dr. R. Kraynak stated that he had reviewed the medical report and pulmonary function study of Dr. Levinson. (EX 6). Dr. Kraynak stated his disagreement with Dr. Levinson's findings on pulmonary function testing. The deposition testimony of Dr. Kraynak was also taken on September 26, 2003. (CX 6). Dr. Kraynak testified that the Miner had continued to be his patient. Dr. Kraynak reviewed his deposition testimony of October 1, 1999, the report of Dr. Matthew Kraynak, the hearing transcript of November 2, 1999, the Miner's testimony and my prior decisions. He also reviewed pulmonary function testing, validation reports, x-ray readings, as well as the medical reports of Drs. Levinson and Kruk. It was his opinion that the Miner suffered from coal worker's pneumoconiosis and that his respiratory disability was significantly contributed to by his coal worker's pneumoconiosis. Some element was attributable to cigarette smoking and to his tuberculosis, however, the vast majority was the result of his coal worker's pneumoconiosis.

Dr. George B. Goodman reviewed the medical evidence by report dated October 19, 2003, also reviewing x-rays which he found to be negative for pneumoconiosis. (EX 7). Dr. Goodman noted the varying smoking histories which ranged from eleven pack years to thirty pack years. Based upon his review of the records, Dr. Goodman concluded that there was no doubt that the Miner suffered severe and disabling respiratory illness. Two factors contributed to this disability, namely chronic obstructive pulmonary disease and his history of pulmonary tuberculosis. Even though the latter disease was treated appropriately and felt to be inactive, Dr. Goodman found the resultant scarring and anatomic distortion that accompanied the illness can prove severely detrimental to lung function and exercise capacity. Dr. Goodman found no grounds for disputing the original judgment rendered by me with respect to pneumoconiosis and respiratory disability. He found no evidence of coal worker's pneumoconiosis, concluding that the Claimant's respiratory impairment and disability had nothing to do with any illness related to exposure to coal dust during his employment.

Dr. Gregory Fino submitted a report dated October 20, 2003, after his review of the medical evidence and his own readings of x-rays, which he found to be negative for pneumoconiosis. (EX 8). Dr. Fino is board-certified in internal medicine and pulmonary disease. Based upon his review, Dr. Fino opined that the Miner did not have coal worker's pneumoconiosis. Dr. Fino did find a disabling respiratory impairment which showed partial reversibility and was consistent with a smoking history as high as thirty pack years. Reversible obstruction, however, was not consistent with coal worker's pneumoconiosis, according to Dr. Fino. It was also his opinion that the reduced pulmonary function values reflected the Miner's tuberculosis. Given the significant scarring in the upper portion of the right lung and significant volume loss and hyper-lucency in the remaining lung, Dr. Fino doubted that the lung affected by the tuberculosis was significantly participating in the Miner's pulmonary capacity. The changes were those typically seen as a result of tuberculosis. All of the Miner's pulmonary impairment and disability were related to cigarette smoking and tuberculosis. Regardless of the presence or absence of pneumoconiosis, Dr. Fino found that the medical data clearly showed that the Miner's impairment and disability were directly related to cigarette smoking and tuberculosis.

Dr. Renn submitted a supplemental report dated October 20, 2003, after reviewing additional evidence. (EX 7). He found that the Miner had old healed pulmonary tuberculosis with residual scarring, the residua of poliomyelitis, superior cervicothoracic kyphoscoliosis, pulmonary emphysema owing to tobacco smoking, traction emphysema owing to contraction and cicatrisation of the right upper lobe, and asthma. In his opinion, a pneumoconiosis did not exist. None of the Miner's conditions were related to his coal mine dust exposure and the Miner did not have coal worker's pneumoconiosis, medical or legal. The Miner's pulmonary emphysema resulted from his years of tobacco smoking and from the traction of remaining lung toward the contracted fibrotic area of the right upper lobe.

In reviewing the medical opinion evidence of record and weighing it against the x-ray evidence of record, I do not find the medical opinion evidence sufficient to outweigh the finding that clinical pneumoconiosis has been established by means of the x-ray evidence. Thus, and in particular, I find the x-ray readings, when coupled with Dr. Galgon's finding that some of the opacities he observed were consistent with pneumoconiosis are sufficient to overcome the contrary evidence of record.

## Arising Out of Coal Mine Employment:

Next, the Claimant must establish that his pneumoconiosis arose, at least in part out of coal mine employment. See § 718.203 (a). It is presumed that the pneumoconiosis of a Claimant who establishes ten or more years of coal mine employment arose out of coal mine employment. Id. Fifteen years have been established in this case. I do not find that the medical reports which have been previously discussed in detail, and are once again, set forth in detail above, are sufficient to overcome the presumption.

### Total Disability Due to Pneumoconiosis

As it has been conceded that the Miner is totally disabled due to a pulmonary impairment, the sole issue left to be considered is whether that disability is due to pneumoconiosis. Total

disability due to pneumoconiosis requires that pneumoconiosis as defined in § 718.201, be a substantially contributing cause of the miner's totally disabling respiratory or pulmonary impairment. Substantially contributing cause is defined as having a material adverse effect on the miner's respiratory or pulmonary condition or as a materially worsen[ing] totally disabling respiratory or pulmonary impairment which is caused by a disease or exposure unrelated to coal mine employment. § 718.204(c)(1)(i) &(ii). Absent a showing of cor pulmonale or that one of the presumptions of § 718.305 are satisfied, it is not enough that a miner suffer from a disabling pulmonary or respiratory condition to establish that this condition was due to pneumoconiosis. See § 718.204(c)(2). Total disability due to pneumoconiosis must be demonstrated by documented and reasoned medical reports. *Id.* The Benefits Review Board has held that § 718.204 places the burden on the claimant to establish total disability due to pneumoconiosis by a preponderance of the evidence. Baumgardner v. Director, OWCP, 11 B.L.R. 1-135 (1986).

In the instant case, while Claimant has established the existence of coal worker's pneumoconiosis and of total disability, I do not find the evidence sufficient to establish that his disability is due to coal worker's pneumoconiosis. In this respect, and for the reasons set forth in my prior decisions, I find the medical opinions of Drs. Galgon, Levinson, Kaplan, Goodman, Renn, and Fino, all of whom are pulmonary specialists, sufficient to establish that the Miner's pulmonary disability is the result of his old tuberculosis, polio and history of tobacco use. While Drs. Simelaro, Kruk, Prince, M. Kraynak and R. Kraynak opine otherwise, I find their opinions are not as well-reasoned or well-documented and accord them less weight. In so doing, I have also weighed the fact that the Drs. Kraynak are the Miner's treating physicians. Their status as such, however, does not entitle their opinions to greater weight, given the other factors to be weighed in making this determination. It should also be noted that neither are pulmonary specialists.

While Dr. Patel lists pneumoconiosis in his 1996 report, it is apparent that the Miner's cigarette smoking was of greater concern to him. Drs. Simelaro, Kruk and Prince find the Miner to be totally disabled due to his pneumoconiosis, however, I find the reports and deposition testimony of the physicians who find otherwise to be more persuasive, given their ability to satisfactorily explain why other factors, including the Miner's old tuberculosis, history of cigarette smoking and polio were the cause of the Miner's pulmonary condition. I also note that these physicians, and in particular, Drs. Galgon, Levinson, Kaplan, Goodman, Fino and Renn were able to review extensive medical evidence in reaching their opinions. Also significant is the fact that treatment records from Dr. Patel, Chapman and others do not indicate a disability due to coal mine dust exposure, but to those conditions found significant by the physicians who attributed the Miner's disability to tuberculosis, polio and cigarette smoking. Dr. Ahluwalia, in 1998, also finds the Miner's condition to be due to conditions other than coal worker's pneumoconiosis. This is true as well with Dr. Rashid in 1998.

While Dr. Galgon indicated he could not differentiate between the opacities resulting from tuberculosis as opposed to coal mine dust exposure, he was able to adequately explain how he could determine the cause of the Miner's pulmonary condition. Dr. Galgon, in his deposition testimony, persuasively discusses and explains in detail why the Miner's clinical pneumoconiosis does not contribute to his disability, pointing to the blood gas study results as evidence that the Miner could not have significant interstitial lung disease as a factor in his shortness of breath.

Given the significance given to the Miner's past conditions by the pulmonary specialists, I do not find persuasive, the dismissal of the tuberculosis and the classification of the Miner's smoking history as "mild" as made by Drs. Simelaro, Prince, Kruk and Kraynak. While some of these physicians attribute the Miner's pulmonary disability to multiple factors, including coal worker's pneumoconiosis, they fail to persuasively explain how this conclusion regarding the etiology of the Miner's disability is reached.

It is apparent that the Miner's tuberculosis and resulting injury to his lung, his smoking history and polio are substantial contributors to his total disability. The evidence is insufficient to meet Claimant's burden of proof, to establish that his exposure to coal mine dust, also substantially contributed to his disability. Thus, based on the opinions of Drs. Levinson, Galgon, Renn, Fino, Goodman and Kaplan, I find that the Miner's coal worker's pneumoconiosis has not had a material adverse effect on his pulmonary condition, nor has it materially worsened a totally disabling pulmonary impairment caused by a disease unrelated to coal mine dust exposure. In so concluding, I have taken into account all the evidence of record. Accordingly, while the Miner has established a change in condition, and the entire record has been reviewed, he has failed to establish he is disabled by coal worker's pneumoconiosis.

### Entitlement

Because the Claimant has failed to meet his burden to establish that he is totally disabled by pneumoconiosis, he has failed to establish that he is entitled to benefits under the Act.

#### Attorney's Fees

The award of an attorney's fee under the Act is permitted only in cases in which the claimant is found to be entitled to benefits. Section 28 of the Longshore and Harbor Workers' Compensation Act, 33 U.S.C. §928, as incorporated into the Black Lung Benefits Act, 30 U.S.C. §932. Since benefits are not awarded in this case, the Act prohibits the charging of any fee to the Claimant for services rendered to him in pursuit of this claim.

### **ORDER**

The claim of R. C. for benefits under the Act is DENIED.

Α

RALPH A. ROMANO Administrative Law Judge

Cherry Hill, New Jersey

**NOTICE OF APPEAL RIGHTS:** If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the administrative law judge's decision is filed with the district director's office. *See* 20 C.F.R. §§ 725.458 and 725.459. The address of the Board is: Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, DC 20013-7601. Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. *See* 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed.

At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Allen Feldman, Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Ave., NW, Room N-2117, Washington, DC 20210. *See* 20 C.F.R. § 725.481.

If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).